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REVIEW OF NUTRITION RESEARCH

Nutrition and Skin Disease

VOLUME 18 · No. 1
JAN.-FEB. 1957

BORDEN'S REVIEW
of
NUTRITION RESEARCH

Reviews and summaries of research information concerning nutrition and foods. Published bi-monthly, by THE BORDEN COMPANY; officers of the Company: Harold W. Comfort, president; E. L. Noetzel, treasurer; D. T. Orton, secretary. Editorial office: 350 Madison Avenue, New York 17, N. Y.

Nutrition and Skin Disease

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GLOSSARY OF TERMS

Intertrigo — an erythematous eruption of the skin produced by friction of adjacent parts.

Seborrhea — an excessive secretion of the sebaceous glands which collects on the skin in oily crusts or scales.

Miliaria rubra — an acute inflammatory (red) rash, commonly called prickly heat or heat rash.

Xanthomatosis — deposition of yellowish or orange lipoidal material in the skin and other organs.

Psoriasis — a chronic skin disease characterized by reddish patches covered with silvery white scales.

Phrynoderma — toad-like skin; dryness of the skin with increased keratinization of hair follicles (Hyperkeratosis follicularis).

Hyperkeratosis — increased production of keratin—the process of keratinization.

Keratosis pilaris — hard, conical elevations in the orifices of the sebaceous glands.

Ichthyosis — skin is characterized by a dry scaly formation with a fish scale appearance.

Introduction

The average adult is covered and "held in" as it were, by about 1.75 square meters (or about 18¾ square feet) of a tough, yet resilient, double layered structure of skin, which is the body's principal bulwark of defense against the entrance of foreign substances. Next to bone, skin has the greatest density and resistance to penetration.

The skin functions to protect against almost every conceivable physical and chemical stress, and when damaged has a remarkable capacity to restore itself. This reflects its dynamic metabolic activity. Through its sweat glands and cutan-

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eous blood vessels, skin provides the chief mechanisms for heat regulation. A child's first sensations of his external environment are felt through the heat, cold, pain and touch nerve buds in the skin.

The skin reflects the age of the individual by its appearance. It mirrors the emotions, such as the blush of embarrassment, the pallor of fear, the full flush of anger and the cold sweat of anxiety. And we are individualized by skin markings through our fingerprints.

A brief review of the structure and physiology of the skin (1) may be helpful in understanding the changes that take place in skin disease, especially of nutritional origin. The skin is composed of three layers: (a) epidermis; (b) corium (dermis or true skin); and (c) the subcutaneous tissue.

The epidermis is a thin layer averaging about 0.2 mm. in thickness, in which the greatest metabolic activity of the skin is located. From the outside inward, the epidermis is composed of cell layers, i.e. (a) stratum corneum, (b) stratum granulosum, (c) prickle cell layer, and (d) the basal cell layer. Melanocyte cells, responsible for the production of skin pigment, are found in the basal cell layer. The epidermis overlies the corium or dermis in a wave-like pattern. The principal metabolic activity of the epidermis is the production of the protein keratin, which is the chief component of the layer stratum corneum. Keratin, by its toughness, elasticity and resistance to chemical change, provides great protection. It is structurally integrated into the flat, shrunken, non-nucleated dead cells of the stratum corneum. The process of keratinization is, therefore, concerned with the production of keratin and the gradual elaboration and replacement of the stratum corneum from the basal cell layer of the epidermis. This response is continuous and may vary with the stimuli on the epidermis.

There are two kinds of skin appendages derived from the epidermis: (a) glandular, i.e. apocrine and eccrine sweat glands, and sebaceous glands; and (b) keratinized structures, i.e. the hair follicles and hair and the nails (1).

The corium or secondary protective layer supports the vascular bed and peripheral nerves of the skin and the epidermal appendages. The corium is also a large potential storehouse for water, blood and electrolytes (1). This is well exemplified in the individual with edema.

The subcutaneous tissue provides further support, is an excellent heat insulator, a good shock absorber, and a storehouse of calories in the form of fat.

General Relation of Nutrition to Skin Disease

From earliest times, man has associated gloss and sheen of the hair coat and skin of his pets and herds with good feeding practices and good general health (2).

Outstanding results of nutritional deficiencies, both primary and conditioned, are skin and hair changes. Rarely, however, are the skin disorders so distinctive that they permit the diagnosis of a specific nutritional deficiency. The skin responds similarly to a wide variety of stimuli. Nevertheless, the dermatologist has

naturally been influenced to use numerous special diets and vitamins in every conceivable dosage level and combination in the medical managment of skin diseases, especially those of questionable or unknown cause. An almost incredible array of results has been published, ranging from extreme enthusiastic support for the value of vitamins to complete denouncement of their use in the treatment of skin disease. As is usual in such matters, the truth lies somewhere in between.

Dermatoses, which may be features of deficiency states, whether primary or due to conditioning factors, can appear on any part of the body, but for the most part tend to be located over areas of irritation; that is, on the backs of the hands, on the front and backs of the wrists, on the elbows, over the face, around the neck, under the breasts, over the knees and feet, and in the perianal region. These lesions are often bilaterally symmetrical and are separated from healthier appearing skin by a sharp line of demarcation. Great variability may be present in such lesions from patient to patient. They are generally accompanied by burning, itching, and considerable pain (3).

Vitamins

VITAMIN A

The first well-documented association of vitamin A deficiency and various types of dermatitis was made by Fraser and Hu in 1931 (4). Their classical description indicates the following sequence of events in vitamin A deficiency. The skin may first be dry and slightly roughened, after which eruptive lesions appear suddenly over rather localized areas of the body. The usual sites of onset are the anterolateral aspect of the thighs and posterolateral aspects of the forearms. These soon extend to involve large areas of the body surface, especially the extremities. The eruption is generally symmetrical in its distribution and is distinguished by an excessive keratinization. Especially marked is the hyperkeratosis of the hair follicles, which may contain intrafollicular keratotic plugs. These plugs may project from the hair follicles as horny spines, while others are covered with a loosely adherent scale which bridges the included follicle. When these plugs are pressed from the hair follicle, a gaping cavity is left. On the face, this eruption may resemble acne, but differs from acne in that the skin is much drier and there is an absence of pustules. Dryness of the scalp is very common. Fasal (5) reported that this follicular hyperkeratotic lesion of vitamin A deficiency could be confused with the follicular type of ichthyosis. In this latter disease, however, the palms and soles are affected, and vitamin A deficiency is rarely involved. A general change in the color of the skin may be seen in vitamin A deficiency, in that the normal pink color fades and becomes sallow or ashen gray.

Vitamin A has been used in moderate to massive doses in the treatment of many skin diseases, especially those associated with excessive keratinization, particularly involving the hair follicle (6). The treatment of such diseases,

other than vitamin A deficiency itself, has not yielded consistently beneficial results. Large doses of vitamin A, in the order of 100,000 to 500,000 units per day, are of some theraputic value in the treatment of pityriasis rubra pilaris and Darier's disease. Such treatment may provide symptomatic improvement, but is rarely curative.

There is potential danger in massive, long-term doses of vitamin A, particularly in children, where doses of 100,000 to 600,000 units per day may produce toxic manifestations. Sulzberger (7) reported that the intoxication from excessive doses of vitamin A actually give the classic picture of vitamin A deficiency in man, particularly follicular hyperkeratinization. Associated with hypervitaminosis A is found rough, itchy, scaly skin, loss of hair and extensive skeletal disturbances with weak fragile bones.

The excessive consumption of carotene or carotene containing foods produces a marked yellow discoloration of the skin which may be confused with jaundice. This condition clears rapidly on reduction of the carotene intake. Although carotene is a precursor of vitamin A, hypervitaminosis A is not associated with carotenemia.

VITAMINS D, K, AND E

There are no specific cutaneous manifestations associated with vitamin D deficiency (6). Skin changes that have been reported are probably secondary to the anorexia and cachexia that develops from this deficiency. A certain percentage of patients with psoriasis seem to benefit from vitamin D therapy, but treatment with large doses of vitamin D over long periods may produce harmful effects (8).

Vitamin K is an essential factor in the synthesis of prothrombin by the liver and thus plays an important part in the normal mechanism of blood clotting. Vitamin K deficiency, while rarely primary, may often result from secondary or conditioned factors. The deficiency is characterized by a reduction of the blood prothrombin level and various types of hemorrhagic diseases appear. Cutaneous purpura may develop extensively and extravasation of blood into subcutaneous tissues may extend over large areas of the body (6). Providing the liver has not been damaged and hence lost its capacity to manufacture prothrombin, the administration of vitamin K rapidly corrects the hemorrhagic tendencies.

Vitamin E has had no clear-cut demonstration of usefulness in human nutrition and no cutaneous manifestations have been asociated with experimental vitamin E deficiency.

THE VITAMIN B COMPLEX

Those members of the vitamin B complex that have been demonstrated as essential for man are closely associated with each other as component parts of enzymes important to the metabolism of energy yielding substrates. The B complex group of vitamins is generally also present as a group in foods. For this reason a deficiency of one member of the B complex group is usually

associated with a deficiency of other members of the group, and therapy must be directed toward supplying all members of the B complex vitamins that might be missing and not just the one that may be related to the predominant deficiency sign or symptom. Because cutaneous lesions are seen in B complex vitamin deficiencies, various members of this group of vitamins have been used in the treatment of skin conditions. The results obtained are generally inconsistent.

VITAMIN B_1 (THIAMINE)

No specific dermatological condition has been ascribed to the lack of thiamine, although Elsom et al. (9) reported that the skin may show pallor, flabbiness and inelasticity which responds to treatment with vitamin B_1. The chief skin change associated with thiamine deficiency is edema.

RIBOFLAVIN

The symptoms of riboflavin deficiency may be divided into three groups — oral, cutaneous and ocular (10). Two changes are usually seen in the oral lesions: first, a general inflammation of the mucous membranes, especially at the borders of the lips; and second, maceration and fissuring at the corners of the mouth. These may spread up or down for 0.5 cm. or more. This manifestation is known as angular stomatitis. The lips may develop abnormal redness covered by a thin gray coat that later sheds off leaving the lips abnormally red along the line of closure. This is called cheilosis and may be accompanied by shallow ulceration, crusting, and vertical fissures. It must be emphasized that angular stomatitis and cheilosis are not characteristic of riboflavin deficiency and other possible causes for these lesions must be ruled out.

Tongue changes in riboflavin deficiency, characterized by purplish or magenta glossitis, have been reported. The fungiform papillae are said to be principally affected and become flattened and slightly enlarged (11).

The dermititis of riboflavin deficiency is primarily a seborrheic dermatitis with many fine greasy scales on an erythematous base. A fine, scaly dermatitis of the hands, perianal area, perineum and vulva have also been described as accompanying the seborrheic picture (12). Dyssebacia refers to a series of disturbances of the sebaceous glands, characterized by increased oiliness of the skin with associated dermatitis, fissuring, and the production of thick, crust-like material from the orifices of these glands. This is differentiated from the cutaneous lesions of vitamin A deficiency primarily by excessive oiliness. In vitamin A deficiency the skin is excessively dry.

The ocular symptoms that have been reported associated with riboflavin deficiency are photophobia, burning and itching of the eyes, and visual disturbances. There is also seen, usually by slit lamp examination, an extensive vascularization of the cornea of the eye (5). However, this also is a non-specific entity, since it may be present in other conditions.

NIACIN DEFICIENCY

The classical triad of dermatitis, dementia, and diarrhea has long been associated with pellagra, which, fortunately, has been practically eradicated from the United States. The use of nicotinic acid therapy and, more important, the large scale enrichment of commonly used foods with nicotinic acid, has been a most valuable public health measure in this accomplishment. Well balanced diets containing an adequate amount of high quality proteins, such as are present in milk, meat, and eggs, supply tryptophan, which may prevent and cure pellagra (13). Pellagra is generally considered a multiple-deficiency disease, with niacin being specific for the treatment of the gastrointestinal, dermal and mental signs and symptoms. Although there are several types of cutaneous skin lesions associated with pellagra (14), the most common form is that of a chronic cutaneous lesion characterized by thick, inelastic, fissured skin, which becomes deeply pigmented. This pigmentation is particularly pronounced in areas that have been exposed to sunlight. Infection is often superimposed in these lesions. Older pellagrins may have another form of dermatitis in which the skin becomes dried, scaly, inelastic and often "too big for the body contained in it" (14). The dermatological lesions in pellagra occur primarily at the trauma points of the body and in areas that are exposed to sunlight.

The tongue and mucosa of the mouth, in acute cases of pellagra, are a bright red, swollen, often ulcerated and secondarily infected. Such changes may be accompanied by corresponding changes in the mucosa of the vagina and rectum. Dyssebacia, with plugging of the orifices of the sebaceous glands with dry, grayish-yellow material, may also be seen in pellagra (15). The disease responds very well to nicotinic acid therapy or to diets containing ample amounts of animal protein.

Nicotinic acid also has the interesting dermatologic effect of producing a marked cutaneous vasodilation with extensive flushing of the skin. This characteristic is not shared by nicotinamide, which is the form of the vitamin used in most vitamin preparations.

VITAMIN B_6

The possible usefulness of vitamin B_6 in skin diseases was indicated from the early work of Sullivan and Nichols (16) and Antopol and Unna (17). It was observed that rats fed a diet completely free of vitamin B_6 developed a symmetrical dermatitis. Wright (18) has studied the effects of pyridoxine deficiency in a number of dermatoses and reported that certain types of eczema, particularly eczema of the seborrheic type, may improve after having failed to respond to local treatment, when pyridoxine is given intravenously or subcutaneously in rather large doses of 24 to 50 milligrams. Pyridoxine deficiency has been produced in human subjects by the use of the pyridoxine antagonist, desoxypyridoxine. In association with this deficiency there was noted manifestations of a seborrhea-like lesion about the eyes, nasal labial fold and mouth, which extended to the eyebrows and skin behind the ears. Many of these cases of seborrheic

dermatitis responded well, at least temporarily, to the local application of pyridoxine in an ointment base, but no effect was observed when the vitamin was given orally or parenterally (19).

Other Members of the B Complex

PANTOTHENIC ACID

While pantothenic acid has been reported to correct the graying of the fur of experimental animals (20), Williams (21) was unable to find any effect of this vitamin on the graying of hair. There is no evidence that pantothenic acid is useful in treating gray hair in human beings.

PARA-AMINOBENZOIC ACID

Conflicting reports have been made concerning the usefulness of this vitamin as a preventative or cure for graying of the hair. Sieve (22) reported that doses of 100 milligrams twice daily over a period of two months resulted in darkening of the hair in all instances. Others have been unable to confirm these observations. It is evident that these rather huge, unphysiological doses of para-aminobenzoic acid have not been evaluated adequately to reach a valid conclusion concerning their importance.

VITAMIN C

The primary structural change in vitamin C deficiency is a disturbance in the formation and maintenance of the intercellular ground substance. The ground substance, an essential matrix of connective tissue comprising the framework of all organs and structures, may well explain all of the multiple and widespread lesions associated with vitamin C deficiency. Although scurvy is not primarily a dermatological condition, the disease may show secondary skin changes, such as perifollicular petechial hemorrhage, hyperkeratisis around the follicle and stiffening and inelasticity of the skin. Ascorbic acid is also concerned with the metabolism of the amino acids tyrosine and phenylalanine (24), which are precursor substances for the formation of melanin pigments of the skin.

General Comments

Pollack and colleagues have recently reported on extensive and carefully controlled studies on nutrition in the Far East (25), which provides well documented support for the prevalance of cutaneous lesions associated with diets inadequate in riboflavin, niacin, and vitamin A. The subjects of the experiment were Chinese Nationalist soldiers who were fed the usual bill of fare, based principally on rice, vegetables and fish. Biochemical studies on blood and urine of these subjects were correlated with the clinical observations of the presence of signs of vitamin deficiency. It soon became evident that vitamin deficits are significant even in a group of individuals considered well-fed as army personnel. It was noted that approximately 80 per cent of the troops of the Chinese Nation-

alist Army on Formosa in the year 1954 to 1955 manifested signs of ariboflavinosis. Approximately 30 per cent of the troops manifested signs attributable to vitamin A insufficiency. About 20 per cent showed pellagroid lesions of the mouth. No obvious signs of beriberi, protein malnutrition or calcium deficiency were noted.

Enrichment of rice with synthetic B vitamins resulted in remarkable remission in the signs attributable to the riboflavin and niacin deficiencies. The inclusion of yellow sweet potatoes in the diet improved the vitamin A status. After a careful statistical analysis of both the biochemical and clinical observations, it was concluded that a strong relationship exists between riboflavin intake and incidence of angular cheilosis, magenta color of the tongue, scrotal dermatitis, and papillary atrophies of the tongue. There is a high correlation between niacin intake and incidence of filiform and fungiform papillary lesions on the tongue. Vitamin A or carotene intake is inveresly related to follicularization of the chest, arms, back and buttocks. These clinical indicators of nutritional deficiency tended to disappear in the men on the enriched diet.

Proteins

The classical evidence of protein deficiency with hypoproteinemia is nutritional edema. The studies of Keyes and collaborators (26) on experimental dietary deficiency demonstrated that long continued deficiency of dietary protein produced extensive skin changes, characterized by dryness, scaliness, inelasticity, and a gray, pallid appearance; all changes suggestive of old age. A substantial number of these individuals also developed a mild follicular hyperkeratosis which somewhat resembled a vitamin A deficiency. One of the unusual skin changes noted was a blotchy, dirty, brownish pigmentation, which appeared almost anywhere on the body, but was most often seen on the face.

More recently, extensive reports on kwashiorkor from widespread areas of the world indicate that this deficiency disease is associated with a protein deficiency, especially animal proteins. This results in faulty growth, hypopigmentation of the hair and other ill defined nutritional dermatoses. Of interest also is the eczematous skin eruptions and inadequate melanin formation observed in the disease known as phenylpyruvicoligophrenia. In this condition, a metabolic block limits the conversion of phenylalanine to tyrosine, accompanied by the increased excretion of phenylpyruvic acid. This disease was first noted in some feeble minded children and new advances in the treatment have been made by the dietary restriction of the amino acid phenylalanine.

Lipids

Several types of cutaneous lesions have been described as being associated with the long term consumption of low fat diets (27). Eczematous patches develop over various parts of the body. It has also been noted that some adults who have excessive fat losses associated with idiopathic steatorrhea frequently have erythematous eczematous lesions (28). Studies of the blood lipids of eczematous

and non-eczematous infants did not reveal a significant difference in the total content of fat in the serum. It was evident, however, that the amount of unsaturated fatty acids was considerably less in eczematous infants. It was found also that their level of arachidonic and linoleic acids in the serum was lower (29). This observation has encouraged the use of fats and oils rich in these two fatty acids in the treatment of eczema in infants (30).

Rajagopal and Chowdhury (31, 32, 33) have conducted an extensive biochemical and clinical study in India on the disease phrynoderma (hyperkeratosis follicularis) in some 65 cases. They found this disease to be extensive in patients eating low fat diets, 28 gm. per day or less. The disease could be well correlated with the intake of essential fatty acids. The essential fatty acid intake of normal and phrynoderma cases was 8.7 and 4.9 gms. per day respectively. The diet of phrynoderma cases was markedly vegetarian and lacking in animal protein. Although phrynoderma has been associated with vitamin A deficiency, only a few of their patients had clear signs of deficiency of this vitamin. The oral administration of raw linseed oil (0.75 oz. per day) and linoleic acid (4 cc per day) produced clinical improvement in two to four weeks and cures in four to 24 weeks.

Since the early pyridoxine deficiency studies with rats had indicated a relationship between pyridoxine and essential fatty acid metabolism (23), Rajagopal and Chowdhury (32) studied the influence of pyridoxine in essential fatty acid metabolism in phrynoderma. They observed by clinical and biochemical observations that the oral administration of raw linseed oil combined with the intramuscular injection of pyridoxine had an added influence in curing phrynoderma.

Overnourishment and Skin Disease

Undoubtedly overnourishment with associated obesity leads to as many skin disorders as almost any other factor. Certainly a sound dietary program leading to weight reduction is a basic adjunct to the treatment of dermatological diseases in general whenever obesity is present. Obesity with excessive fat folds often leads to intertrigo, which is caused primarily by friction between skin surfaces and by the maceration of the skin resulting from accumulated moisture in skinfold. These changes often lead to infections, particularly with staphylococcal organisms or to the fungus diseases (dermatophytosis and monoliasis). In addition, obesity may activate a latent diabetic tendency with all of the accompanying dermatoses associated with this disease. Obese people, because of their thick subcutaneous layer of fat, tend to become overheated easily and sweat more profusely. This produces undesirable effects on normal skin, exaggerating areas of inflammation and producing skin rashes. Prickly heat or miliaria rubra is a commonly recognized evidence of sweat retention.

Special Diets

Almost every possible variation of diet composition and dietary restriction has been used in an attempt to cure some of the different dermatoses. Acidifying

and alkalinizing diets, dehydration, low carbohydrate, low fat, low protein, low caloric, high caloric, high protein and high fat diets have all been employed at one time or another (6). A very careful analysis of such studies leads one to the conclusion that there is little evidence of their value. Some of the more practical dietary suggestions are (6): (a) restriction of carbohydrate consumption in the presence of chronic staphylococcal infection. The exact reasons for the beneficial effects of such a diet are not clear. (b) It has often been suggested that diets which contain large amounts of carbohydrate and fat have an undesirable affect on acne vulgaris. Again, it is difficult to find consistent substantiation of this idea. (c) Hypercholesterolemia may be reduced by the dietary restriction of fat and/or the consumption of sitosterols, to minimize the development of hypercholesterolemic xanthomatosis.

In general, it appears that a well balanced diet with a moderate to high intake of protein plus supplemental vitamins when indicated, and total caloric adjustment to prevent excessive obesity is the most rational dietary plan in the management of dermatoses.

Allergy

Food allergy may be the cause of many skin lesions, including atopic dermatitis, urticaria, and infantile dermatitis and eczema. The importance of nutrition in pediatric allergy was extensively reviewed in a recent issue of *Borden's Review of Nutrition Research* (34).

The capacity for a food to become an allergen depends on such factors as the nature of the food, the quantity and concentration of the food in the diet, and the duration of exposure to the food. In addition, one must add the predisposing factors that make up the individual's constitution. An allergic family background should be considered an important danger signal indicating special care in the feeding of infants and small children, particularly during the period of "physiologic, immunologic immaturity." For example, Glaser and Johnstone (35, 36, 37) have reported that when soybean milk was substituted for cow's milk or human milk at birth, there was a marked reduction of infantile eczema, other milk allergies, and a decrease in the development of later unrelated allergic phenomena.

Since food allergens enter the body primarily by way of the gastrointestinal tract, derangements of the "G.I. system" are among the prime factors leading to allergy. This is predominantly related to the gastrointestinal absorption of unaltered food protein.

Neurogenic and psychogenic factors have also been implicated as associated causal agents in allergy. These relationships are as yet little understood.

Almost all foods and beverages have at one time or another been cited as allergenic. Those that are most often associated with allergic reactions are as follows (not necessarily in order of allergenicity): unaltered egg protein, milk

proteins, fish and seafood, wheat, corn, tomatoes, spinach, cabbage, asparagus, rhubarb, celery, onion, garlic, and citrus juices and fruits.

The importance of early diagnosis and correction of allergy, especially in infants and children, cannot be overemphasized. Often, the diagnosis is made on the basis of a high index of suspicion. Skin tests with food allergens and elimination diets are employed to track down the offending food or allergenic substance.

When the allergenic role of one or more foods has been established, the intolerance can be handled by: (a) elimination of the food in question, or by (b) specific desensitization. This is carried out by oral or parenteral administration of small and gradually increasing doses of the allergen. The problem of food elimination may lead to rather bizarre dietary patterns, and caution must be exercised to be sure that adequate and balanced nutrition is maintained by appropriate substitution.

Discussion

The cardinal principle in the treatment of disease is to find the cause and remove it. This emphasizes the importance of an accurate diagnosis as a prelude to therapy. Skin diseases produced by exogenous means, such as contact dermatitis from chemical substances, irritation from trauma, or excessive exposure to heat or sunlight, are usually managed with ease. There is no reason to consider nutritional therapy under these circumstances other than to make certain that dietary practices consistent with good health are enforced. The dermatoses produced by metabolic or emotional aberrations on the other hand, may often present a real challenge, both in diagnosis and in treatment. If disease is believed to result from flaws in metabolism governed by intracellular enzymes, it is obvious that food must play a significant and fundamental role in the proper functioning of these metabolic processes. On this basis, it is entirely conceivable that less than optimal nutritional practices, if imposed for long periods of time, may well provide the basic mechanism for a deranged metabolism that ultimately leads to a dermatological disease. These are conditions which, in general, respond very slowly, if at all, to any kind of therapy. Since so little is known about the metabolic alterations resulting from chronic malnutrition, it is little wonder that the rather haphazard use of vitamins, minerals, and special diets have produced few consistent results in dermatological problems not clearly associated with deficiency states. Usually the cutaneous signs of clinical malnutrition are non-specific and may be produced by many other diseases which must be ruled out before a direct nutritional cause can be related to the skin abnormality. It must also be emphasized that while the acute lesions of malnutrition are rapid in onset and quickly responsive to therapy, chronic lesions, such as the cutaneous lesions associated with primary or conditioned malnutrition, are in general produced slowly and respond slowly to therapy. Metabolic insults suffered over a long period of time are not likely to respond to therapy overnight.

Research must be increasingly directed toward a better understanding of the metabolic factors which produce dermatologic disease before rational therapy can be applied.

Any consideration of skin disease would be incomplete without mention of the importance of the psyche. Certainly skin blemishes can give rise to many personality and emotional problems. Who can assess the psychological trauma suffered by "the kid with pimples?" It is most difficult to evaluate whether anxiety produces skin disease or skin disease produces anxiety. Greatly expanded interest has been evoked in the potential relationship between the metabolism of the central nervous system and psyche by the recent development and use of tranquilizing drugs.

Summary

There is little question that dietary deficiency, whether primary or conditioned, is associated with dermatological lesions which clear on therapy with the proper nutrient. These lesions have been particularly associated with vitamin A, riboflavin, niacin, vitamin C, vitamin K, and pyridoxine. Dermatoses associated with obesity make it clear that careful dietary restriction in these cases is important. Undernutrition, a much more rare phenomenon in this country, may be associated with general pallor, scaling, loss of hair pigment, dryness and loss of elasticity of the skin. Allergy to foods may often cause skin disorders and such foods should be eliminated from the diet and substitutions made to maintain good nutrition. With the exception of the use of vitamin A in large doses, which has provided occasional benefits in the rare skin diseases of pityriasis ruba pilaris and Darier's disease, there is little indication for the therapeutic use of vitamins in dermatology conditions, unless clear-cut evidence of associated primary or conditioned deficiency can be demonstrated. A careful nutritional history, physical examination, and, if necessary, biochemical studies should be conducted to rule out evidence of nutritional abnormalities. The underlying defects related to dermatoses of unknown etiology may be the result of improper nutrition and metabolic error endured over very long periods of time.

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Printed in U.S.A.